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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

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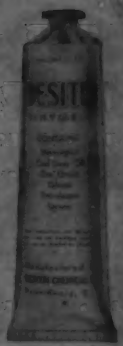

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(1) Wilson, J. L., and Dickinson, D. G.: J. A. M. A. 158: 261, 1955.

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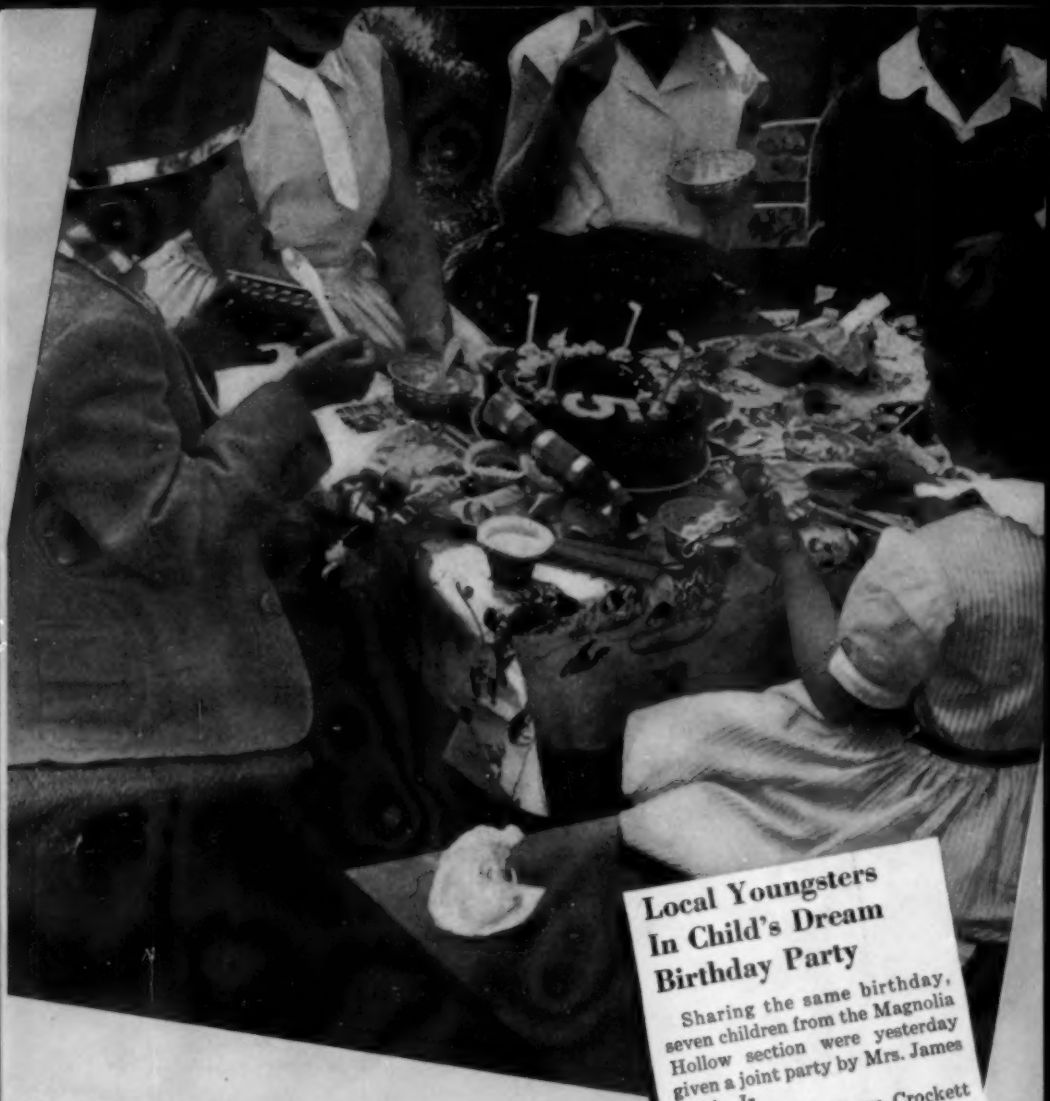

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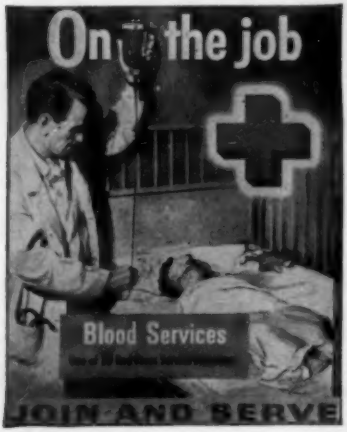

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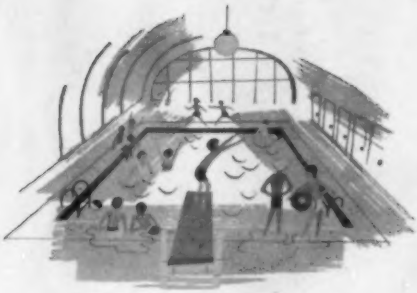

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Vol. 73 APRIL 1956 No. 4

JOHN FITCH LANDON, M.D., Editor

CONGENITAL ACROMICRIA SYNDROME*

HYMAN GOLDSTEIN, M.D., Sc.D.
New York.

The congenital acromicria syndrome, heretofore termed "Mongolism", describes the condition and symptomatology of children who are mentally retarded to a greater or lesser degree, and, who resemble the normal Mongolian child in appearance. This illness is one in which an anomalous pattern of growth occurs during intra-uterine fetal development. In rare instances the condition is not recognized for several weeks after birth. It is important that the parents be told about the condition by the attending physician as soon as a diagnosis is made so that proper medical care, and parental guidance and care, can be given to the baby at the earliest possible date. The prognosis is so much better when treatment is begun early.

The term "Acromicria" was brought to us by Schuller[1] in 1907, by Clift[2] in 1922, by Benda[3] in 1949, and the terminology of "Congenital Acromicria with Mongoloid Features" by the author[4, 5] in 1954. "Acro," means end-point, while "micro" means small. This corresponds to the underdeveloped ends of the long bones and the partially dwarfed extremities of the affected children. I suggest the name of "Congenital Acromicria Syndrome" in place of "Mongolism" because it is more adaptable to the pathogenesis and symptomatology of the condition, and does not carry, suggest, or bear with it any stigma for the child.

Clinical research, based on a study of 200 cases of congenital acromicria syndrome covering a period of 30 years, brought to light many prenatal conditions and influences, tracing the etiology to

* Lecture delivered April, 1956 at the University of Panama, Republic of Panama.

noxious conditions and, an exhausted and weakened maternal reproductive organism within the expectant mother which causes an abnormal growth pattern of the developing fetus in utero. What are the prenatal conditions and influences?

1. *The Mother's Age:* 79 per cent of the mothers of congenital acromicria syndrome children were from 30 to 40 years of age when pregnant with them. I consider this the risk age range of pregnancy. The youngest child in a large family of siblings is the most likely one to be affected. In over 25 per cent of the mothers there was a history of having had a sterile period of more than five years before the birth of the abnormal baby. Proper child spacing, preferably between two and four years is a good margin of safety. This etiological factor suggests that fertilization of a dormant ovary may affect the normal fetal pattern of development diverting it to an abnormal one.

2. *Miscarriage:* 39 per cent of the mothers have had one or more miscarriages. This is a very high index when we compare it to the maternity hospital reports of the general population which is less than 1 per cent. Most of them have had menstrual difficulties as to periodicity, character of the flow and pain. This is due to an imbalance of the bodily hormones during pregnancy. Its adjustment is essential for better fetal development. Evan Shute,[6] in analyzing bio-chemical serum changes during pregnancy in uterine bleeding, interrupted pregnancy and fetal malformations, found an excess of blood estrogenic hormones, diminished amounts of progesterone, and deficiency in vitamin E, and blood serum trypsin-like enzyme. Normally, during the end of the first, and the middle of the second trimester of pregnancy, Krebs and Bartlet,[7] found that the fetal pancreas secretes trypsin to help replenish the mother's trypsin drain in order to digest the excess estrogens of the blood and prevent uterine bleeding and other complications. As the progesterone rises during this period, the urine gonadotropin diminishes.

3. *Contagion:* Two mothers contracted German measles, and one measles. Viruses and some bacteria can filter through the placenta and attack the fetus.

4. *Heredity:* This is questionable in considering it as a direct factor in causing congenital acromicria syndrome. I have had only one case of identical twins in whom only one child was affected and the other normal. Dr. Benda,[8] in a study of 500 cases of

acromicria syndrome or mongolism, concluded that heredity was of no importance as a factor in its etiology. Macklin[9] in 1950, reported that of eleven identical twins, ten of the twins were both congenital acromicria syndrome cases, and one twin a normal and an affected child; while out of thirty-nine fraternal or dissimilar twins only three pair were identical, and thirty-six pair were of one normal and one affected newborn. This implies the presence of a necessarily genetic factor that may expose the fetus to a congenital acromicria pattern of development, when there is an environmental factor or factors operating, in which the mother fails to protect the fetus from its genes. It is also interesting to note the characteristic, minute skin ridges of the hand, which form definite patterns, and which are the basis of every governmental identification system also are the subject of scientific interest. These patterns take form during the third to fourth months of fetal life and remain unchanged during life. The Simian or Macacus palm furrows, such as the furrow which crosses the palm from the side of the thumb, meets the flexion furrow line which starts beneath the little finger, and ends between the second and third fingers. They are always separate and, therefore, never meet in the normal hand. The Simian or Macacus palm furrows are common in about 60 per cent of the affected children and in one or both hands of their parents. Cummins,[10] Evatt[11] and Bonnevie,[12] reported these findings. From a genetic viewpoint, the possibility of a mother giving birth to another congenital acromicria syndrome child following a previous affected sibling is slim.

5. *Diet:* The pregnant women's daily diet must be a proper one. This has a great bearing on the pediatric rating of the expected newborn. 90 per cent of the mothers of congenital acromicria syndrome children had a daily intake of less than 60 grams, in some instances as low as 35 grams, protein in their diet. There was also a deficiency in the caloric, vitamins, and mineral intake. Dieckman[13] and coworkers reported a definite and striking correlation between the incidence of abortion and protein daily intake. While abortions occurred in 8.11 per cent of the women consuming less than 55 grams protein daily in their diets, and 3.96 per cent in those with a daily intake of 75 to 85 grams protein, it was practically nihil in the subjects who consumed more than 85 grams protein daily. They reported also a high index of excellent pediatric ratings for

babies among 302 infants examined, born to mothers who were on a high daily protein intake. This was confirmed also by Dr. Weston A. Price,[14] who made an extensive nutrition research study among the primitive natives in isolated jungle country. He found their foods to be very rich in protein content, vitamins and minerals. The natives were in unusually excellent health and their skeleton and muscles well developed and strong. Congenital acromicria syndrome was unknown among their siblings. In other isolated jungle countries that were modernized by visiting soldiers during the first world war, by fur trappers and by tourists who mingled with and traded with the natives, bartering refined white flour, sugar, candies, all kinds of canned goods and jams, trading them for pearls and trinkets, resulted in most of the natives eating these modernized foods as delicacies in their daily diets. In a short period of time, all kinds of diseases of physical degeneration popped up among the men, women and children of the population, and congenital acromicria syndrome newborn became common in their midst.

The congenital acromicria syndrome child suffers from an anatomical and physiological growth deficiency with varying degrees of mental retardation. The child's forehead and skull appear large proportionately to the smallness of the face because of the undeveloped middle third of the facial skeletal structure, i.e., the maxillary, nasal, vomer, ethmoid, and sphenoid bones. The sphenoid, paranasal and frontal sinuses are absent or rudimentary. The eye-sockets are oval, small and almond shaped, pointing up and outwards giving the child a mongoloid appearance. Internal strabismus, lateral nystagmus and blepharitis are common. Occasionally we encounter cataracts and other complications. The anterior fontanelle is usually large and the sutures may be found widely separated, but which close after much delay. There is a lack of cartilaginous development and its reaching the end plates at the epiphyses, especially during the first three to four years, and again between puberty and adolescence. This leads to a deceleration of the growth of the long, short, flat, and irregular bones. We observe in them shorter extremities, pudgy short hands and feet, incurved little fingers, small facial bones and, short stubby sphenoid as seen by x-ray. The bones are short and wide. The occiput is flat making the anterior-posterior skull diameter shorter by 2 to 4 cm. than

the normal. We are concerned here with a small cranial brain case in a child, which cramps and compresses the brain, pituitary gland, and the brain stem, thereby interfering with their growth, expansion and functions. The small cerebellum is pushed forward. The cerebrum is found flattened and compressed in all of its lobes. The cerebellum is small. Although the original patterns of the fissures and convolutions are not disturbed, the different lobes of the brain are pressed against the cribriform plate, and, lobe against lobe so that they appear mushroom in shape. The pia and arachnoid membrane may adhere to the cortex. The Purkinje cells may be found arrested or absent in several cortical layers. Demyelination of the white matter or neuroglia is striking. Clusters of new blood capillaries and small vessels are sometimes found in the temporal area. The structures of the brain and its functions, and the body and its functions are extremely immature. It takes an untreated child about ten years to mature to the equal of a two to four year mental age. Brain metabolism and oxygen supply is disturbed and lowered as are also brain functions of its cells and the neuroglia. It is interesting to note the results of the research by Himwich and coworkers,[15] who found the normal brain in children to consume 14.2 mgm. carbohydrate from each circulation through it, while the retarded child's brain consumes from 35 per cent to 50 per cent less as it draws its supply from the blood upon which the brain depends for its fuel and energy. The infant's brain normally takes up 8.59 volume per cent oxygen, while the retarded child's brain takes up only 3.63 volume per cent oxygen from the blood stream. This is a reduction of 57.7 per cent, corresponding closely to the percentage of mental retardation.

Normally, the brain weighs 350 grams at birth, 800 grams at one year, and 1350 grams at maturity, a gain of 450 grams the first year, and only 50 grams a year thereafter to maturity. The kidneys together weigh 25 grams at birth, 65 grams at one year, and 250 grams at maturity; the heart is 25 grams at birth, 40 grams at one year and, 250 grams at maturity; the thymus gland is 10 grams at birth, and 30 grams at puberty and diminishes in size and functions from then on; the spleen is 10 grams at birth, 25 grams at one year and 150 grams at puberty and may be palpable upto 3 years of age; the liver is 150 grams at birth, 300 grams at one year and, 1500 grams at maturity. The circumference of the head is 13

inches to 14 inches at birth; 17 inches to 18 inches at one year; 18 inches to 19 inches at two years; 19½ inches to 20½ inches from three to five years; and 20½ inches to 21½ inches at maturity. Note the marked gain, in size and weight, during the first year of the brain, as well as all the other organs, and, the circumference of the head. This is true also of the height and weight of the body during the first year, and even the second year of life as compared to the relative slowness of gain and body development thereafter. It is evident that if the children affected with congenital acromicria syndrome are neglected and left untreated during the first two years, they suffer a severe set-back in brain, tissue and other organ maturity and development. Let us compare a two-day old brain and organs from a full term baby who died on the second day after birth from congenital acromicria syndrome reported by Dr. Benda,[16] with the normal figures. The weight and size of its brain equalled that of a seven-month premature. The liver weighed 44 grams, the normal is 150 grams; the spleen was 4 grams, the normal should be 10 grams in weight; the kidneys weighed 10 grams as against 25 for the normal, and the heart and blood vessels were proportionately smaller and lighter. The report of a brain and organs of a four year old child showed similar findings with flattening and crowding of the brain lobes, small cerebellum and abnormal vascular changes of the brain. The brain convolutions and fissures are intact but distorted by compression from the smallness of the brain case or child's skull. The mental retardation, emotional and other responses are proportionately affected depending on which areas and lobes of the brain are compressed the most. Demyelination of neuroglia and immaturity are prominent with the brain gray and white matter. The immature character of the spinal cord must be kept in mind since we find here evidence of insufficient myelination of the nerve fibers, vascular stasis, and spongy edema of the white matter which accounts for some of the poor muscular tone and poor coordination we find in these cases. Laboratory findings in cases of congenital acromicria syndrome reveals low blood fat soluble vitamins, which deficiency is found in the blood of the mothers during the pregnancy of the affected ones. Vitamin C is also found deficient. The newborn is affected as the baby's blood sodium, calcium, thyroid hormone, reticulocytes and lymphocytes are somewhat lower, while the blood cholesterin, and esters,

gammaglobulin, figrinogen, eosinophiles, chlorides, and total granulocytes are increased. These findings give us additional leads in research follow-up work, and in our therapeutic approach. There are roughly speaking about 3000 congenital acromicria syndrome newborn in the United States each year. It is, therefore, of great interest to us to do what we can to help them, and also, in a prophylactic way, to prevent their occurrence. Because of the character of the abnormal fetal growth and developmental pattern in utero of these cases, we find a general pluriglandular underdevelopment, with either the pituitary gland dysfunction predominating as in the more severe types of acromicria congenital syndrome, or the thyroid glandular hypofunctioning predominance in the milder cases of mental retardation. These major parent glands of the body, mainly affected, influence most of the functions of the other ductless glands to share in the abnormal clinical findings.

There has been an increasing number of children who suffer from congenital acromicria syndrome (mongolism) in Panama City and in the Republic of Panama in recent years. The physicians attending the Hospital del Nino, the hospital for children in a separate building of the Hospital Santo Tomas, Panama, Republic of Panama, became very much interested in this problem, especially Dr. Hermelinda de Varela, now President of "La Sociedad Panamena de Pediatria," who wrote asking me to help them. On December 13, 1955, I received an invitation from Dr. Edgardo Burgos, President, at the time, of the Pediatric Society of Panama, to visit Panama, hold clinics and lectures to enlighten the clinics, pediatric, psychiatric and psychological, as well as the social service staffs on how to diagnose, treat and cope with these children. On March 7, 1956, I received an invitation from the Secretaria, Mr. Diogenes Arosemena of the Universidad de Panama to deliver a series of three lectures in the auditorium of the University. In the interum, Dr. Hermelinda de Varela was elected President of the Pediatric Society of Panama. The invitation from the University of Panama was supplemented by a joint interest and invitation from the University of Panama and the Pediatric Society of Panama; the Panama government, through the Minister of Social Work and Public Health, and the Director of Public Health of Panama, through Dr. Hermelinda de Varela invited my wife and myself to make the trip to Panama and be their guests from April 1 to and

including April 21, 1956. This article is my first lecture delivered, on April 3, 1956 in the University of Panama Auditorium. Two other lectures delivered on April 6 and 9, 1956 in the University of Panama Auditorium, and one lecture delivered April 19, 1956 to the Pediatric staff at the Hospital del Nino in Panama, and one lecture delivered at the National Medical Association of the Republic of Panama April 14, 1956 will be written up and published in future issues.

While in Panama City, I held daily clinics with the Pediatric Staff at the Hospital del Nino and enlightened the attending physicians in the endocrinological changes and the symptomatology and diagnosis of congenital acromicria syndrome, and allied encephalopathies presented by me each clinic day. The therapy for each case was thoroughly discussed. I saw more than fifty cases within two weeks. I found the work very pleasant, very constructive, and the clinic personnel and the doctors most sincere and co-operative, and stimulating to work with. Before leaving, I helped organize and establish a Retarded Children's Clinic in the Hospital del Nino, a Parent Association to Help the Retarded Children, and a kindergarten school for retarded children where, besides the teacher and parent's help, the children are guided and cared for by the psychiatrist, psychologist, psychological social worker and the pediatric staff of the Hospital del Nino Children's Retardation Clinic supervised by Dr. Hermelinda de Varela. I held a conference with the principal and teachers of a twenty classroom public school, making many suggestions to them on how to cope with the retarded children; how to revitalize and reorganize their work in preparing a practical program in association with competent medical and psychological staff personnel to teach the school aged, exceptional children in whom the Government of Panama is now showing great interest. The conference also was a very pleasant, cheerful and co-operative one. I was promised that this school will be given over to educate the educable and trainable children. I also arranged for the parents of the exceptional children to hold weekly meetings, several of which have already been held at which a psychological social worker, acting as moderator, officiates and listens to individual reports from the mothers about their children during the previous week, and remedial suggestions are made. At these meetings, the mothers hear about the successes and the failures, the

behavior problems of each child, and learn the approach and corrective methods each mother uses, the reasons for the same, and explanations obviated by the moderator. I received, recently, correspondence indicating that the work is progressing successfully. I am very happy to report on these activities in Panama, Republic of Panama, and I feel confident the exceptional children will benefit, their parents will be happier since a brighter future for both is ahead.

I wish to express my sincere appreciation and my gratitude to the Minister of Social Works and Public Health of Panama, the Director of Public Health of Panama, for helping to create public school classes for the school age retarded children; to Dr. Hermelinda de Varela, President of the Pediatric Society of Panama, and to Dr. Edgardo Burgos, Past President of the Pediatric Society of Panama for their help; to Dr. Rolando Chanis for his scholarly interpretation of my lectures into Spanish; to the members of the Panama Pediatric Society, the members of the pediatric staff of the Hospital del Nino, and the medical staff of the Hospital Santo Tomas, as well as to the social service staff and Medical Superintendant of the Hospital del Nino for their sincerity and cooperation in organizing the children's retardation clinic, in which Dr. H. de Varela played an important part, and in which all worked with me. My thanks and appreciation to Mr. Diogenes A. Arosemena, Secretary of the University of Panama, for the use of the University Auditorium and for his kind cooperation and invitation to come to Panama and hold the Lecture Conferences there on April 3, 6 and 9, 1956. Many thanks also to the parents of the exceptional children for their help in organizing an association to help the Retarded Children of Panama and in establishing a kindergarten nursery for them, and also to the principal and teachers of the Panama Public School for the Retarded Children, newly established there.

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ANTILEPTOSPIRAL EFFECT OF MILK. L. Kirschner and T. Maguire. (New Zealand M. J., 54:560-564, Oct. 1955).

Leptospirosis is prevalent in man and animals in New Zealand, but, at variance with other parts of the world where rodents present the reservoirs of human infections, domestic farm animals (pigs and cattle) are the predominent sources in New Zealand. Of 262 patients diagnosed in the laboratory, 200 (80%) were dairy farmers or members of their families. The rest were all in some contact with livestock. It is known that cattle after an acute or subclinical stage of disease may excrete virulent Leptospira with the urine for several months. As in brucellosis the infected urine could serve as a source of human infection through the milk. So far, however, no milk-borne infections have been reported from the many countries where bovine leptospirosis is known to be widely spread. The authors found that undiluted milk rapidly killed Leptospira. The studies they describe in this paper were concerned with the distribution and nature of the antileptospiral principle in milk. They made studies on 100 samples of cow's milk, 20 of human milk, 10 of human colostrum, and 10 of goat's milk. They found a destructive factor against virulent and nonvirulent mutants of Leptospira. It was present in milk from all three species with some variations in titer. The principle is mainly associated with the casein. In the whey the titer is much lower. The mode of action (rapid dissolution of the organisms) by this factor in milk and its physical properties are similar to, but not identical with, lysozym. This natural protective agent in milk explains the absence of milk-borne infections in the many countries where bovine leptospirosis is widely spread.—*J.A.M.A.*

A NEW TOPICAL REMEDY FOR THE PREVENTION AND MANAGEMENT OF NAPKIN DERMATITIS (DIAPER RASH)*

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AND
H. H. SAWICKY, M.D.

Following publication of the paper by LeVan et al.[1] on the use of silicones in various dermatoses, including uncomplicated "diaper rash", we were stimulated to investigate further, the preparation reported, or a modification thereof, in the prevention and management of the "diaper rash" complex.

Napkin dermatitis or "diaper rash" has plagued infants, mothers and physicians for generations. Dermatologists and pediatricians are in general agreement that the designation "diaper rash" is a nonspecific term to denote an eruption in the napkin area of the infant; that it is not a disease entity and that it may be caused by varying known and some unknown etiologic agents. Amongst the known causes are ammonia dermatitis, intertrigo, contact dermatitis, atopic dermatitis, seborrheaic dermatitis and monilial dermatitis. All of these entities are aggravated by lack of cleanliness, wetness, maceration and secondary bacterial infection.

Materials. An effective topical remedy or prophylactic agent, therefore, is one that incorporates a moisture repelling agent, a keratolytic and a bactericidal agent. Such a cosmetically acceptable medicament† was prepared containing the following ingredients:

1. Silicones (Dow Corning 200 or 555), 3 per cent.
2. Glyoxyl diureide, 0.2 per cent.
3. Hexachlorophene, 0.25 per cent.

incorporated in a highly emollient ethanolamine stearate base. Known sensitizers, possible primary irritants and antipruritic agents, no matter how low their sensitizing index, were deliberately withheld from the emulsion. The efficacy of incorporating a

*Aided by a grant from and material supplied by the Pharmacal Division, Revlon, Inc., New York, N. Y.
†Baby Silicare®, a product of the Pharmacal Division, Revlon, Inc.
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moisture repelling agent in the management of ammonia dermatitis has been demonstrated.[2] The ability of the dimethyl siloxane polymers to repel moisture, while themselves remaining inert, nontoxic and nonsensitizing has prompted their widespread acceptance as dermatologic agents[3, 4, 5, 6, 7]. For over half a century, the diureide of glyoxilic acid has been used as an adjunct in topical therapy. It is a stable and nonirritant keratolytic which stimulates epithelial healing[8, 9, 10]. Inhibition of the growth of ammonia forming bacteria has been reported to be of benefit in the reduction of inflammatory processes associated with diaper rash[11, 12, 13, 14]. Hexachlorophene, an effective and relatively nonsensitizing bactericidal was selected as the best available degerming agent.[15]

Toxicity and sensitization studies have been reported by others[1, 16, 17]. Recent work has revealed that the silicone preparations are efficacious in the management of bedsores.[7, 18]

CLINICAL EVALUATION

The subjects of this study were divided into three groups. The first group consisted of seven hundred and twenty-six newborns followed in the hospital nurseries. Of this group, four hundred and twenty-nine were treated with the test product and two hundred and ninety-seven with products normally used in routine hospital care, as the control group. All the newborns, test group and control, were cared for under identical hospital routine procedures and were checked daily for signs of skin irritation, however mild. The results are charted in Table 1.

TABLE 1

	No of Cases Studied	No. of Cases Free from Eruption Throughout Study	Skin Reactions Noted	Percentage of Reactors
Test Product	429	408	21	4.8%
Controls	297	265	32	14.1%

The second group consisted of one hundred and ten infants, ranging in age from one to eighteen months. They were followed for a period of six months, being observed at intervals of two to four weeks during the entire test period. Mothers in this group were given the following instructions:

1. To make no change in the general care of the infant.
2. To avoid the use of all medicaments, i.e. (oils, lotions, oint-

ments, powders), other than the prescribed emulsion. This was applied to the napkin area two to three times daily.

3. To cleanse the diaper area with lukewarm water following urination and defecation.
4. To report immediately any evidence of a dermatitis or untoward reaction.

Of the one hundred and ten infants studied, eighty-three were free of any eruption on the initial examination and remained so during the entire period of the study. Five, who originally showed no sign of a dermatitis, developed a mild erythema in the napkin area during the period of observation. The emulsion was discontinued until the erythema had subsided and then reused on four of these infants without a recurrence of the erythema. The fifth infant was lost to the study. The remaining twenty-two subjects presented varying degrees of napkin dermatitis, ranging from an erythema to an intertrigo, to a mild papulovesicular eruption at the original examination. Twenty of these twenty-two cleared completely on the medicament, one cleared partially, and one remained unchanged. Severe vesicular, bullous or weeping dermatoses were not included in the study. The results in this group are charted in Table 2.

TABLE 2

No. of Cases	Appearance of Napkin Area on Initial Examination	Results	Remarks
83	Clear	Clear	.....
5	Clear	Mild erythema	1 lost to study; 4 stopped emulsion until cleared, then reused it and remained clear.
22	Erythema, intertrigo, mild papulovesicular eruption.	20 cleared completely; 1 cleared partially, 1 unchanged.	No complications

The third group of subjects presented the common dermatoses associated with infancy, as charted in Table 3. Again, severe vesicular bullous or weeping dermatoses were not included in the study. The emulsion was applied to the involved areas three times daily. No other medicament was used but rubber and/or plastic panties were avoided.

Incidental observations made during the course of the study were of interest. Some mothers used the preparation on the faces of infants with "saliva eczema" due to drooling and finger sucking,

TABLE 3—*Results*

Diagnosis	No. of Patients	Clear	Partially Clear	Unchanged
Intertrigo	16	16		
Napkin area erythema	17	15	2	
Atopic eczema	2	1		1
Contact dermatitis	2	1	1	
Bedsores	1	1		
Total	38	34	3	1

and reported excellent results. Other mothers and nurses remarked on the efficacy of the emulsion in keeping their hands smooth and soft.

SUMMARY

A new emulsion for the prophylaxis and management of napkin dermatitis is described. Five hundred and seventy-seven infants, ranging from newborn to eighteen months of age were studied with gratifying results. The medicament is cosmetically acceptable, pleasant to use for both mother and child, efficacious, and relatively free from side reactions.

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INFANTILE HYPERTROPHIC PYLORIC STENOSIS IN PARENT AND CHILD. T. McKeown and B. MacMahon. Arch. Dis. Childhood, 30:497-500, Dec. 1955).

One hundred and twelve living patients in whom the diagnosis of pyloric stenosis was confirmed by operation at the Birmingham Children's Hospital in the period 1920-1934 were traced in 1955. They had 29 children, none of whom had pyloric stenosis. By combining these results with those from two reported series, the incidence of the disease in children of affected parents is estimated as 6.9%. Ten cases of pyloric stenosis in parent and child were obtained from current records of children treated at the Birmingham Children's Hospital. By inspection of records of 2,579 patients treated in hospitals in Liverpool, Manchester, Newcastle-on-Tyne, and Sheffield, two more examples of the disease in parent and child were found. In eight of the 12 families, the affected parent was the mother. When the results are combined with those of two published series, there are 33 families, in 17 of which the mother was affected. Since pyloric stenosis is much more common in males than in females it is concluded that the risk of the disease is considerably greater (at least four times) in children of affected mothers than in children of affected fathers. It is also shown that the risk of pyloric stenosis is not spread uniformly among all offspring of parents who have had the disease, but is highly localized in certain families. Among children in families in which a parent and one child have been affected, the proportion exhibiting the disease is approximately 40%. The authors feel that these results are inconsistent with a simple genetic hypothesis and are more plausibly attributed to the influence of the early postnatal environment.—*J.A.M.A.*

PEDIATRICS AT THE TURN OF THE CENTURY

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

INTUSSUSCEPTION IN INFANCY AND CHILDHOOD*

A Collection of 1,028 Cases With Statistics

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In presenting a paper on so general and interesting a subject, it becomes necessary to select a few of the main topics for discussion to the exclusion of the remainder, except in so far as they are illustrated by the reports of my 3 cases, which are as follows:

Case I. Baby N., aged eight months, breast-fed, had diarrhea for several days, recovered spontaneously, and was well for several days prior to present illness.

Present History. May 2, 1904, had a natural bowel movement about midnight; began to vomit at 5 A.M. Vomiting and crying at intervals until seen at 10 A.M. Enema at 10 A.M., followed by some bloody fecal matter, and flatus; this was followed by straining, crying, and passing of bloody, watery stools.

Physical Examination. Fairly well nourished, pupils moderately dilated, reacted to light. Mouth and throat, nose and ears negative. Tongue slightly coated and dry; chest negative; abdomen moderately distended and soft, more resistant on right side of median line. On palpation, some gurgling over the small intestines: an elongated, resisting mass could be palpated from region of cecum following course of ascending, transverse and descending colon to about an inch below the left costal arch; this was slightly movable in every direction, considerably so from its terminus in the left hypochondriac region. No tumor could be felt per rectum, but the finger returned covered with bloody mucus. Temperature, 99.6°F.; pulse, 140; respiration, 40. The child was sent to the hos-

*Read before the Chicago Medical Society, February 8, 1905.
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pital and was operated upon two hours after the first examination.

Operation. Anesthetic, chloroform; time, twenty minutes. On entrance to hospital the stomach was washed out, with return of considerable mucus. Enema given returned with great amount of small clots and some fecal matter. A median incision two inches in length was made above umbilicus. The tumor, which was about ten inches in length, extending from the splenic flexure to the cecum, was reduced without traction, except the cecum, which was brought up into the wound. The cecum and appendix were highly congested and edematous, but were returned without further manipulation, and the wound was closed. Temperature after operation, 100.2°F.; pulse, 130; respiration, 36.

Second Day. Temperature rose to 103.8°F.; pulse, 138; respiration, 64 at 3 P.M. Water had been given in one-half teaspoonful doses, and one teaspoon of breast-milk at 12, 1 and 2 P.M.; the child vomited shortly after taking each dose with rise in temperature. All feeding except chipped ice was stopped and one-eighth grain doses of calomel and soda were given for two doses, also an oil enema. At 10 P.M. child passed a large amount of flatus.

Third Day. Temperature rose to 103.2° F., pulse, 134, respiration 66 at 2 P.M. Three doses of calomel and soda were given, followed by citrate of magnesia; no food administered except water; child vomited repeatedly throughout the day, and after five doses of magnesia passed considerable flatus.

Fourth Day. Afternoon rise of temperature 103° F., bowels moved seven times between 1 and 10 P.M., breast milk again being given. Fourth to eighth day, temperature ranged from 100° to 103° F., bowels moved regularly and child continued to improve.

Tenth Day. Stitches removed. Twelfth day left hospital. Has had no illness of any kind since.

CASE II. Raymond M., age three years; history negative, except for fact that he has been accustomed to stand on the rear rod of a three-wheeled bicycle and to lean against the seat, in which way he is pulled around the house.

Present History. April 3, 1905, 8:30 A.M., sudden pain in abdomen, violent in character. Mother says it was so severe that baby stood on his head and she could see a tumor-like mass to the right of the umbilicus; about 10 A.M. began to vomit, returning all food and water, of the latter of which he drank a great deal.

Enema contained considerable fecal matter, seen by the writer at 1 P.M.

Physical Examination. Negative except abdomen. Abdomen flat, no tympanitis, an indistinct resistance noticeable in right hepatic region and this part of the abdomen was the most sensitive. The child allowed a thorough examination to be made without crying, only pushing the hand away when it reached this part of the abdomen. Temperature 98.6° F. per rectum, pulse 120, respiration 32, rectal examination negative. At 9 P.M. the findings were the same, except that the mother had given an enema at 4 P.M., which she said returned slightly tinged with blood and contained no fecal matter; there was still resistance in hepatic region but no distinct tumor palpable, no typmpanitis. Temperature 99° F., pulse 120, rectal examination negative.

April 4, 1905, 8 A.M., twenty-three and one-half hours after first pain, slight tympanitis, resistance still noticed but no distinct tumor. Temperature 99.4° F., pluse 130, rectal examination negative. Enema given under pressure of three feet returned no fecal matter, but a few bloody mucus pieces about size of appleseed.

Operation. Twenty-six and a half hours after onset. Total time forty minutes, incision two and a half inches through right rectus; some free peritoneal fluid, tumor mass lying in region of cecum and extending to hepatic flexure, reduced by pressure from below upward without traction, was easily reduced except last six inches, which was enteric and was decidedly edematous, infiltrated and had it serous surface covered by fibrinous adhesions. This loop was dark blue in color, but circulation was restored by hot compresses; a slight serous tear necessitated three small silk sutures. The invagination began in the ileum as an enteric intussusception about eight inches above cecum; after reduction bowel was replaced and omentum brought down to cover it; abdomen closed in four layers. During operation child wrapped in blankets and surrounded by hot-water bags.

Subsequent History. Vomiting, which had occurred every ten to thirty minutes before operation, ceased; three hours after operation water was given in teaspoon doses every fifteen minutes. Next day one grain of calomel was administered in one-tenth grain doses, followed by three one-half ounce doses of citrate

of magnesia; this was followed in about twelve hours by passage of flatus and shortly after by fecal matter. Nourishment in the way of liquid peptonoids and beef tea were started the day following operation; peptonized milk on second day. With exception of daily fluctuation of temperature between 100° F. and 102½° F., recovery was uneventful and child left hospital on fourteenth day.

CASE III. Baby G., age four months, breast-fed. On February 23, 1904, history of uneasiness and frequent desire to go to stool. Slight frequent stools, and occasional vomiting since February 20th. During morning of 23d, began to pass bloody mucus and the mother called a physician for first time .First seen at 2 P.M. Child pale, listless, skin moist and pulse rapid, constantly grunting, passed some bloody mucus from bowel while being examined. The abdomen was flat and a tumor could be felt in the sigmoid region, extending upward. Child taken to hospital and operated upon two hours later.

Operation. Anesthetic, chloroform; time, about twenty minutes. Tumor extending from cecum to sigmoid region was rapidly reduced and abdomen was closed without further manipulation. Before operation temperature was 104.2° F. per rectum; at 8 P.M., 103° F., pulse 126, respiration 60. Child was given 1/100 grain morphin and a little water.

Second Day. Temperature ranged between 100 1/5° F. and 102° F., pulse 100 to 160, respiration 32 to 48. Child took water but refused breast; sharp, piercing cries, breathing seemed painful, passed flatus at 1 P.M.

Third Day. Temperature rose to 106° F., pulse 120, respiration 64. Bowels moved three times during day; child grew continually weaker until 4 P.M., at which time it died.

Autopsy. Heart negative, left-sided bronchopneumonia. At the site of the intussusception some exudate; bowel thickened and hemorrhagic, but no recurrence of intussusception.

PATHOLOGY (MICROSCOPIC)

In presenting the microscopic findings of our case, one of the ileocecal variety, which was probably one of that class of cases most favorable for surgical interference, and which was operated on within eighteen hours after onset of the first symptoms of invagination. I desire to emphasize the necessity for early

surgical interference if we hope for a lowered mortality in this rapidly destructive lesion of the bowel. The mucous membrane and its underlying submucous coat seemed to have borne the brunt of the destructive process in both the ileum (section one-half inch above the ileocecal valve) and the colon (section one inch below the ileocecal valve), which formed a part of the intussusception. The mucous membrane showed areas of marked infiltration, destruction of the glandular elements and several areas of ulceration of the mucous membrane down to the submucous coat and occasionally involving the muscular layer of the bowel. The submucous layer was the seat of hemorrhagic infiltration, separation of its constituent fibres, with a resulting thickening of the entire coat, in places being from four to eight times its normal thickness. The seat of the round cell infiltration was most marked beneath the ulcerated areas in the mucous membrane. The lymphadenoid tissue forming the solitary follicle and Peyer's patches was greatly increased. In my specimen, in which the peristaltic action of the bowel had been restored, the infant dying on the fourth day following operation from a bronchopneumonia, the circular and longitudinal muscular layers, as well as the serous coats, had undergone little change.

The right lung was the seat of a bronchopneumonia quite generally distributed throughout the middle and lower lobe. The great lesson taught by the unfortunate outcome in this instructive case, I believe, is the necessity for early and radical surgical treatment, though we must remain in doubt as to whether the source of the pulmonary infection was through the respiratory tract direct or by infection taking place through the ulcerations in the bowel. That every hour of delay means an increased danger of absorption of intestinal bacteria, with probable infection in distant organs even when the muscular and serous coats remain intact, while with their involvement the dangers of general or a local peritonitis are only too imminent.

D'Arcy Power reports the various histologic changes exhibited by the portions of intestine involved in an intussusception, in a series of 31 cases, from which I quote. The result of his examination shows that any part of the intestinal wall may be affected, but that one portion usually suffers more than others, and the stress of the affection falls most often upon the submucous tissue

and upon the circular layer of muscle. The mucous membrane, too, may be seriously injured, but the longitudinal layer of muscle and the serous coat are the least often affected. The earliest histological changes are correlated with an effusion of blood, but the amount of the extravasation varies greatly, at one time so slight as hardly to displace the tissues, at another time so considerable as utterly to destroy them. The site of the extravasation also varies. It may be in the mucous membrane—and it seems that this occurs in the most acute cases. It is usually in the submucous coat, though it may be in the muscular layers of the serous coat. The extravasation is followed by inflammatory changes, in which the submucous tissue and the circular layer of muscle are chiefly involved. These changes terminate in a hyperplasia of the connective tissue leading to sclerosis, in a tryptic (pancreatic) digestion, leading to the disappearance of every cellular element in the wall of the bowel, and the conversion of its connective tissue into reticulin, in diffuse suppuration or in sloughing of the inflamed bowel, which is then separated and cast off by the ordinary process of ulceration.

ETIOLOGY

The cause of spontaneous intussusception is unknown, but D'Arcy Power has shown that the width of the large intestine at birth is only a few millimeters greater than that of the small intestine. Before birth its diameter is the same, or even a little less, while at the age of fifteen years it is two and one-half to three times as large. The colon begins to grow in girth directly after birth, though it remains for a time almost stationary in length. The ileum, on the other hand, grows both in length and breadth. The ileum, however, rarely doubles its diameter in the course of its growth, but the large intestine not only often doubles its size, but may even treble or quadruple it. These facts seem to have an important bearing upon the question of the origin of intussusception in young children. The colon is growing in width rapidly and continuously from birth onward, but at about the age of four months, the exact time when spontaneous intussusception becomes common, it also begins to grow in length. The small intestine has grown steadily in length and breadth from the beginning, though the increase in its circumference is less rapid than the increase in

its length. During the early months of a child's life, therefore, there is a rapidly increasing disproportion between the transverse diameters of the large and small intestines and physiology teaches that too rapid growth is often associated with perversion of the function, especially when, as in this case, the increased rate of growth affects both the muscular and the nervous tissues. Unduly rapid growth of the large intestine may even allow the end of the ileum to become prolapsed into the colon, and, under suitable conditions, such a prolapse may serve as the starting point of an intussusception. When an intussusception has once been started, the anatomic peculiarities of the individual alimentary tract are of paramount importance, for they determine the character of the intussusception. In the ileocecal forms the colon, with few and simple ileocolic folds devoid of lymphatic glands, will allow the intussusception to run a chronic course even though the amount of bowel invaginated is very great. Complex fossæ with numerous lymphatic glands at the ileocolic angle and prolongations of mesentery along the wall of the ileum will, no doubt, so far steady this portion of the small intestine as to render its invagination less likely, though, should it occur, the additional amount of tissue invaginated will render the impaction peculiarly tight, so that if gangrene be not produced at once, early adhesions will be formed and the intussusception will soon become irreducible.

The first event in the formation of an invagination of the bowel is an energetic annular tetanic contraction of some portion of the intestine. This contracted area constitutes the fixed point from which the invagination develops. The invagination, however, is not produced in such a manner that the contracted portion of the bowel is forced into the normal intestine situated below this spot by the peristaltic waves coming from above, on the contrary, the invagination is primarily produced by the action of the musculature of the normal portion of the intestine situated below the contracted spot. It is probable that the longitudinal muscles of the intestinal wall of the piece of intestine are chiefly active, and that they pull the normal intestine situated below the point of spastic constipation upward over the contracted piece of bowel.

When the process of invagination has once started, and particularly when it exceeds physiologic limits, it is enforced and reinforced by the same factors that initiated it. It is quite pos-

sible that the head of the invagination, after it becomes tightly wedged into the sheath, constitutes the primary irritant which causes further spasmodic constriction of portions of the bowel situated above it, or causes violent peristaltic movements of this portion of the bowel, which, in its turn, carries the incarcerated head downward just as it would propel any other constituent of the bowel contents onward.

In reviewing the literature of the past ten years, I have been able to collect 1,028 cases, reported in a total of over 200 articles; some of these cover a period previous to 1894, but all have been reported since that date, and with the exception of some of Gibson's cases, and a few others, all have occurred under the age of sixteen years. Some of the reports of groups of cases are necessarily incomplete, but in the 314 cases which I have individually collected, and are reported either singly or in small groups, I have been more fortunate in getting details.

Direct Causative Factors. Although the anatomy of the bowel is undoubtedly a very important factor in the development of a spontaneous intussusception, in a considerable number of cases we have a history of an exciting factor. Foremost of these are intestinal disturbances in children, which may or may not secondarily cause a local paresis of the bowel with overaction of a neighboring segment. Lichtenstein believed this to be one of the most important factors. He found 21 cases in which diarrhea preceded intussusception out of 593 cases, while Fitch found it in 13 out of 45 cases.

Others direct factors as recorded in my series and those of others are as follows:

Number of cases		314
No factor recorded		272
Constipation		3
Digestive disturbances		5
Blow on abdomen		3
Indigestible food	Boiled peas	1
	Cherry stones	1
Round worms in small intestines		1
Papilloma of cecum		1
Lymphosarcoma of intestine		1
Long mesentery of cecum, 7 inches and 9 inches		2
Invagination of Meckel's diverticulum		6
Invagination of appendix		9
Tubercular ulcer of cecum		2

Nontubercular ulcer of cecum	1
Tubercular mesenteric lymph nodes	2
Enlarged postcecal lymph nodes	4

Fitz in his series states that the exciting causes were absent in 42 cases and the following were the possible causes in 45 cases:

Diarrhea	13
Habitual constipation	12
Protracted abdominal pain	7
Indigestible food	6
Violent exertion	4
Injury	3

Lichtenstein's series are many in adults, but suffice to say that of 593 cases, in 267 there was an absence of a history of the cases and in 111 it developed suddenly in healthy individuals.

Hirschsprung reports 64 cases. One-third of these cases were absolutely healthy up to the time of onset. The majority were suffering from some intestinal disorder. In 14 diarrhea of over ten days' standing preceded the strangulation. In others constipation had been troublesome.

Age is certainly a most important factor, and one on which Power lays particular stress. My series illustrates the predominance of this lesion in the first year of life. Cases in which the age was stated numbered:

1-3 months	8	
3-6 "	75 = 23 per cent	= 60 per cent
6-12 "	118 = 37 per cent	= 60 per cent
1-2 years	18	
2-3 "	12	
3-4 "	13	
4-8 "	32	
8-18 "	24	
Not given	14	

The two youngest, aged six days each, both died, irrigation only being tried. The remaining five under three months of age, respectively aged eight, seven and eleven weeks, were operated with recovery of the first and last. The remaining two were not operated and died.

In Gibson's cases, 81 were one year or under, and 49 ranged from one to ten years.

Hirschsprung reports 64 cases, of which 46 were under one

year, 9 in the second and 9 from two to eight years.

His youngest case was forty-nine days, and of the 46 under one year, 39 were breast-fed exclusively (85 per cent), only 2 were bottle-fed from birth, and 13 had received other food than milk.

SYMPTOMS—SKETCH OF THE CLINICAL PICTURE

The suddenness of the onset is the most striking characteristic of this condition. The remaining symptoms may vary directly or indirectly according to the degree of the strangulation of the intestinal and mesenteric circulation, and the permeability of the intestinal lumen, and in enumerating them I shall attempt to classify them according to their diagnostic importance. We find a rapid and unexpected development of a train of symptoms reaching their maximum intensity within a short time, more often in a perfectly healthy child, though not infrequently we have a history of some intestinal disturbance or more rarely one of abdominal trauma; they may appear while the patient is at rest, in motion, during feeding, or when asleep. In the majority of cases, the first symptom noted is a sudden violent pain of a colicky character, not infrequently appearing to radiate from a definite point; this is usually shortly followed by vomiting. These two symptoms may be considered to be a constant occurrence in young children. At this time the child usually has one or more bowel movements.

These are usually at first diarrheal in character; later, though not invariably, mucus, blood and mucus, or pure blood, may be passed together with thin liquid bowel contents. At this time, or even earlier, symptoms of marked prostration are invariable and may soon be followed by collapse; the pulse becomes small and rapid, the attacks of vomiting usually recur and may become feculent, by which time usually there is no passage of fecal matter by the anus, though some bloody or mucous material may be evacuated. Tenesmus is frequently a source of great suffering, more especially after obstruction of the bowel has become complete. A rise of temperature in the early stage is rarely observed and the same may be said of advanced degrees of meteorism.

I. *Abdominal Pain.* This is the first symptom of acute invagination. Its onset is without premonition, colicky in character, usually uninterrupted at the onset, later becoming intermittent;

its location varies with the seat of the intussusception. But in children this latter point is of little value, because of their inability to localize it.

II. *Nausea and Vomiting.* Nausea and vomiting occur either simultaneously with the pain or immediately after. In my collection of 314 cases, in those in which this symptom is noted, vomiting is recorded as being present in 166, absent in 4 cases—the first following a blow on the abdomen, the second a double intussusception comprising a descending ileocecal and an ascending colic; the other two were of the ileocecal variety. All recovered. Four cases had a record of fecal vomiting, two of which recovered: one by irrigation on the first day; one by laparotomy on the eighth day. Of the other two one died without operation and the other was subjected to a circular enterorrhaphy with a fatal result. In 1 case the vomit was bloody, with recovery.

In 52 cases reported by Martin, vomiting occurred in 89 per cent. Fitz reports its presence in nearly nine-tenths of his cases on the first day, and fecal vomiting in 12 out of 93 cases appearing on or after the fourth day, in all but 2 of his late cases. It may be continuous or occur at intervals; the higher up in the intestinal canal the invagination has occurred the more prompt and constant will be the onset of emesis. The earliest vomiting as seen on the first day may be regarded as reflex.

III. *Evacuations of the Bowels.* In acute cases we usually have one or more evacuations of fecal matter, which may vary from thin liquid to formed stools, and represent the intestinal contents below the obstruction. After this has passed we have a complete absence of all fecal matter and flatus if occlusion is complete. After the congestion of the intestine becomes more marked and inflammation of its walls begins, we have passages containing blood, serum and mucus. Hemorrhagic evacuations represent one of the most constant symptoms of invagination, present in 156 of my cases in which the history was detailed and absent in only 4 cases. The amount of blood varies from a few streaks to a profuse hemorrhage which may cause death. When the condition becomes subacute the hemorrhagic evacuations may cease transiently or permanently until destruction of the bowel has taken place, when they may again become bloody, contain gangrenous intestinal wall and have a characteristic odor.

IV. *Prostration.* Prostration sudden in development and out of proportion to the other symptoms present, especially when associated with great pain, little fever and a moderate degree or absence of tympanitis, should lead to the suggestion of a possibility of intussusception.

V. *Tumor.* The tumor of invaginations is the most important physical sign from the diagnostic standpoint. In 197 cases in which there is a complete history of the case recorded in my collection, 183 give a history of the presence of an abdominal tumor and an absence in 14 cases, with the presence of a rectal tumor in 35 cases and absence of same in 38 cases. In 11 there was an absence of abdominal and a presence of rectal tumor. Martin records presence of a tumor in 79 per cent out of his 52 cases. Hemmeter records presence of a tumor in 308 out of his 610 cases. Lichtenstein reports presence of a tumor in 222 out of 433 cases. Raffinesque found it in 24 out of 53 cases of chronic invagination. A tumor may exist and still be too small to be palpated. This is especially true of enteric intussusception. Location of tumor is variable. According to the table of Lichtenstein, the most frequent seat is the region of sigmoid flexure. In my series, 10 cases were in right iliac region, 13 right hypochondriac region, 14 region of transverse colon, 7 in left hypochondriac, 12 in region of descending colon, 24 left iliac region, and 13 in the region of the umbilicus, out of 94 cases in which location was stated. Invagination tumors are relatively very movable, though in rare cases with chronic course they may become fixed and immovable by adhesion.

VI. *Meteorism.* The tympanitic distention of the abdomen depends on the degree of obstruction of the intestinal lumen, upon seat of invagination, and upon the presence of diarrhea. Meteorism is usually late in developing and its absence is of diagnostic import.

VII. *Tenesmus.* Tenesmus is much more frequently present than is meteorism, being especially severe in intussusception of the sigmoid region and the rectum. Martin reports its presence in 77 per cent of his cases.

VIII. *Condition of the Abdomen.* Aside from the comparatively rare tympanitic distention already spoken of, there are usually no characteristic symptoms or signs, recognizable on the

abdomen superficially. In exceptional cases we recognize the site of the tumor by an elevation as described by the mother in my Case II. In enteric intussusception, there is usually an elevation in the region of the umbilicus.

IX. *Fever.* Fever occurs in about 40 per cent of all cases of invagination in which the symptom is referred to, early in the attack. Its presence is to be expected when complications have taken place.

DIAGNOSIS

I will not attempt to go into differential diagnosis at this time. Phosphorus poisoning, opium poisoning, undescended testicle, appendicitis and thrombosis of mesenteric artery are instances of cases seen by the author which required differentiation from intussusception.

Whenever a child who has previously been in good health, or giving a history of intestinal disturbance, presents the group of symptoms—sudden onset of abdominal pain, bloody stools, slight fever, and a prostration out of proportion to the other symptoms —the possibility of an intussusception should be thought of. These combined with nausea and vomiting are almost a constant picture seen in this condition, and when seen together with tenesmus, suppression of fecal evacuation and flatus, our picture needs only to be completed by the absence of, or a moderate degree of tympanitis, allowing us to feel the presence of an elongated abdominal tumor. And none the less important is the palpation of the tumor per rectum. The diagnosis of a chronic form in the absence of an intussusception tumor presents great difficulties, and in these cases it is of especial importance to most carefully and minutely study the history of the case.

PROGNOSIS

The course of intussusception in the great majority of cases in adults is acute, in the small minority it constitutes a chronic disease, in infancy and childhood the disproportion is even greater. Experience teaches that the fatal termination is the most frequent and therefore the prognosis is grave, because of the many factors which we encounter and which tend to delay the proper therapeutic procedures necessary to a successful outcome in their treatment.

Rapid in onset, more rapidly progressive, they demand a properly directed, judicious and radical treatment. I believe that the prognosis depends to a great extent upon the individual treatment of each case, which calls for a most careful consideration not alone of the patient and conditions he presents, but also of the hygienic surroundings and the preparation of the physician to give his patient the most modern methods at his command.

In summing up the data which I have at hand, I hope to produce convincing evidence of the lowered mortality which goes hand in hand with the advent of improved surgical technique and its early application in cases of intussusception.

Of a total of 1,028 cases collected, 314 are cases reported singly or in smaller numbers throughout the literature of the past ten years, and these I have attempted to classify more in detail: 211 recovered, 103 died—of these 34 recovered under non-operative treatment, 26 died; 83 were operated and recovered after operation, previous to which a number of attempts had been made to reduce by irrigation, gas, etc., while 36 died under this method of treatment; 69 were cured by laparotomy after one or no attempts to reduce by irrigation, so far as can be ascertained by the histories given, while only 5 cases reported under this method of treatment died. In the remainder the details of treatment were not stated. Of this group of cases we find that 43 per cent treated by non-operative method died, while 30 per cent of operative cases following several attempts of irrigation died, and only 8 per cent of the cases reported following a single or no attempt at irrigation died, showing that the earliest operation with the least manipulation gives by far the best results. Even though we know that many of the fatal cases are not recorded in the literature, thereby apparently lessening the percentage of mortality, which is apt to be misleading, we are still impressed by the great difference in the percentage of mortality, 8 per cent in the earliest operations and 30 per cent in the later.

Of the total number of 38 resections recorded, 21 were fatal and 17 recovered; of these 23 were done by suture with 9 recoveries; 10 by the Murphy button with 4 recoveries; one by the bobbin with recovery, and the case of Dr. Jacob Frank with resection of the cecum in a seven months infant with recovery. In this case the union was made by the Frank bone coupler.

Artificial anus was made in 9 cases with 4 recoveries; while in the remaining series of 714 cases collected, of 71 resections, 18 recovered, and of 24 cases of artificial anus, 5 recovered. A most interesting group of cases are those of the small bowel, of which I have a record of 22 out of 314 cases of intussusception, with 11 deaths and 11 recoveries. Of the 11 deaths, 1 was operated on the second day requiring resection, due to gangrene; 8 cases on the third day, 6 of which were gangrene, while the 7th presented adhesions; 2 cases on the fourth day, with adhesions and gangrene. Of the recoveries, 4 were operated on first day, 2 on second, 1 on the fourth, 1 on the fifth, and in the remaining 3 the day of disease was not stated.

When we realize the impossibility of trying to reach the seat of an enteric obstruction without operation we certainly have a strong argument for early operation without manipulation of these cases. Some of the interesting points noted in this series of cases and which affected the prognosis are:

1. Spontaneous sloughing of intussusception with recovery (1 case).
2. Recurrence after apparent reduction—After irrigation 3 deaths and 4 recoveries, after operation 1 death and 1 recovery. Showing small percentage of recurrence after operation.
3. Invagination irreducible—2 recoveries by resection, 8 deaths.
4. Retrograde intussusception—1 with recovery by operation.
5. Bowel in 1 case incised to allow escape of gas, with recovery.
6. Second invagination not found at operation, 2 deaths.
7. Peritonitis—8 deaths.
8. Pneumonia—1 recovery, 2 deaths.
9. Tears in peritoneum, with suture—3 recoveries, 2 deaths.
10. Rupture of bowel during reduction—2 deaths.
11. Shock after operation—4 deaths.
12. Convulsions—3 deaths, 1 recovery.

TREATMENT

We now come to sum up intussusception in its practical aspect, dealing with its treatment, which is of as prime importance to the general practitioner as to the surgeon, for on the former depends the disappearance of the old classification into—first, incarcerated, and second, strangulated forms. In the great majority of cases,

neglect of treatment alone carries the bowel to strangulation and is also responsible for the majority of irreducible cases.

For our further consideration we must treat all cases on the basis of their being reducible or irreducible cases.

History of Treatment. The earliest works of special note on this subject were probably those of Ashhurst, 1874, Lichtenstein, 1873, and Jno. Hutchinson, 1874, although both injections and abdominal sections have been employed for centuries for the relief of this condition. The abdomen was occasionally sectioned in the days before modern surgical technic was discovered, but usually later in the disease with the inevitable result of a suppurative peritonitis and its lethal issue.

Laparotomy in intussusception received a distinct impulse in the eighties when Braun in 1885 and others revived the surgical treatment and more especially advocated early surgical interference.

Irrigation in Intussusception. Clinical study of a series of cases especially as evidenced by the older authors before surgical interference was safe, has taught us that there is an inherent tendency toward spontaneous reduction before the time of paralysis of the muscular coat and formation of adhesions have rendered it impossible. And it is before decided pathologic changes in the intestinal wall have taken place that we may hope to obtain results by irrigation or other mechanical methods of reduction. Experiments by Mortimer, in 1891, upon the unopened bodies of children, for the most part under two years of age, shows that there was apt to be a cracking of the serous coat of the large intestine when the resultant pressure of the fluid distending the colon is about two and a half pounds, that is to say, when the irrigator is raised five feet above the patient, and this accident usually happens when the irrigator is raised eight feet. Mole arrived at substantially the same results, and as he worked with the abdomen open, he was able to see the exact manner in which the intestinal rupture occurred, as a result of its over-distention. When this accident is imminent, the peritoneal coat of the bowel splits longitudinally for a considerable length; the fluid then begins to leak through the wall of the gut, a small jet issues, and at last, if the pressure be continued, a large rent takes place with forcible expulsion of the contents of the bowel in the peritoneal cavity. Rupture of the large intestine is most likely to occur in the transverse colon at or near the splenic flexure, whilst in the small intestine it takes

place in the unprotected portion of the bowel which is situated between the two layers of mesentery.

Mechanical distention should be done under complete anesthesia, combined with gentle external manipulation of the abdomen; the surgeon should be present, and be prepared for incision if irrigation proves unsucessful, or in case of accident, so that there may be no further delay. If these conditions cannot be met, except under extraordinary circumstances this method of treatment should not be practised. After apparent reduction by this method the child should be kept under constant observation for recurrences, as incomplete reductions frequently occur and are an indication for immediate surgical procedure.

Capacity of the Colon. It is impossible for the surgeon to esti- in which irrigation is likely to be successful in an ordinary case of intussusception, nor can he judge the amount of pressure which may be applied with safety to the inflamed and softened intestinal wall at the neck of the tumor.

Method of Irrigation. Forty-eight hours is the limit of time within which irrigation is likely to be successful in an ordinary case of ileocecal intussusception with acute symptoms, and in most cases far less than this. And such pressure is alone justifiable in a child of two years, as can be obtained by raising a reservoir of water containing a quart of salt solution at 100°F. two and one-half feet above the anesthetized patient; long continued distention under low pressure is of more avail than rapid dilatation under a high pressure and is far less likely to kink the bowel and thereby prevent the pressure reaching the seat of the invagination. Gentle kneading from below upward aids the irrigation, as possibly does also the inverted position described by Jacobi, with child on its abdomen resting on a soft pillow with hips elevated. The length of an intussusception is no bar to its reduction by irrigation, for many cases are recorded in which an intussusception has protruded beyond the anus. The duration of the symptoms is perhaps always of less importance in an intussusception than their intensity, for a long standing intussusception is often more easily reduced by irrigation than one of comparatively short duration. The longer the time the symptoms have lasted, however, the more likely it is that adhesions will have been formed. Slight adhesions are not an insurmountable barrier to reduction by irrigation, though they militate greatly against its success.

Contraindications to Irrigation. Abundant hemorrhage would seem to contraindicate any attempt to reduce the intussusception by irrigation. Much extravasation of blood implies destruction of the muscularis mucosæ, infiltration of the submucous tissue, edema of the circular muscle, and consequently a swollen condition of the mucous and submucous layers, with paralysis of the muscular coat. The swollen tissues render reduction difficult, and if the intussusception be reduced, the paralysis of the muscle allows recurrence to take place, and may thus lead to the loss of much valuable time. Absence of hemorrhage, on the other hand, associated with severe collapse, equally contraindicates the treatment of intussusception by irrigation, for it points to the early occurrence of gangrene.

After-treatment of Cases Reduced by Irrigation. The after-treatment of an intussusception which has been cured by irrigation must consist in keeping the patient absolutely at rest, in the administration of opiates, and in feeling the abdomen gently from time to time to ascertain that the tumor has not recurred. The following are the disadvantages of intussusception:—

1. Impossible to gauge the amount of pressure.
2. Impossible to exclude presence of serious changes in the bowel wall or the more complicated forms of invagination.
3. Impossible to ascertain when reduction is complete.
4. Injuries of bowel during irrigation heighten the mortality of laparotomy.
5. Delays surgical interference.

Treatment by Abdominal Section. When invagination after one or more trials under proper conditions has failed to reduce the invagination or only relieved it partially, or there is a suspected recurrence, this method of treatment should in all cases be discontinued and abdominal section performed.

Pitts, in British Medical Journal, 1901, reports 49 cases occurring between 1897-1900, in which all except 1 case were treated primarily by abdominal section, the exception being a twenty-four hour case in a seven months infant with resulting cure. He reports twenty-seven deaths and twenty-one recoveries in the remaining 48 cases, a marked lowering of mortality at the St. Thomas Hospital over preceding years. This radical procedure is due to the fact that, in his experience, cases which came to the surgeon have previously been subjected to irrigation repeatedly and this has only too infrequently been done in conjunction with the internal admin-

istration of purgatives, which combination has already created a tendency toward collapse.

To Prevent or Minimize Shock. 1. Place child upon a hot water bed or bag.

2. Envelop extremities and chest in cotton, wool or some equally serviceable protective.

3. Administer a minimum of anesthetic.

4. Operate with rapidity and caution, with the least possible exposure and manipulation of viscera, protecting them where possible by hot sponges. This can often be facilitated by making first a small incision, which can be easily enlarged as necessity requires. Another procedure which can be employed profitably in a considerable number of cases, especially those along the transverse and descending colon, is the partial reduction by warm water pressure per rectum just previous to operation, in this way reducing the size of the incision required.

A median incision, beneath the umbilicus, in most cases, answers, but the rectus incision is undoubtedly better, Erdmann's advice on this point being of value. He finds that in most cases going through the right rectus is best, except when the tumor is found in the region of the descending colon or sigmoid flexure. He further states that he has never found it necessary to stitch the gut or mesentery to the parietes for the usually ascribed cause of long, lax mesentery or mesocolon, believing that sufficient temporary adhesions will form as a result of congestion and edema of the gut that is finally extended from the intussusceptions. Shortening the mesentery in cases where there is grave doubt as to the reduction remaining permanent is the simplest procedure.

The method of attempted reduction is of initial importance. Never pull on the entering or proximal end, but use pressure on the apex of the mass through the bowel walls, from below upwards; this will avoid much of the danger of tearing the bowel coats. Slight adhesions can often be broken up by a blunt director applied between the layers, and reduction be accomplished.

Where there is any difficulty in the final reduction, or the condition of the bowel is uncertain, the same should be completed outside the peritoneal cavity. Any serious tears of the serous coat of the bowel should either be remedied by suture or covered by omentum, and suspicious bowel should be treated by such methods as the case suggests.

Difficulties in replacing inflated bowel are of very frequent occurrence, especially in young infants. In such cases the incision should be enlarged early, and not after several vigorous attempts at reduction. By this means they can usually be replaced. Where such a solution is impossible incision or puncture of the bowel may become necessary. With the bowel distended in this manner every precaution should be taken not to include the bowel in the abdominal wall sutures.

TREATMENT OF IRREDUCIBLE INTUSSUSCEPTIONS AND THOSE CASES IN WHICH THE BOWEL IS MORIBUND

Congestion and the loss of the bowel's natural gloss, which is so frequently seen in the severer and older cases of invagination, should not cause the surgeon to too hastily assume that the bowel is dead, but rather lead him to test its vitality by pricking it gently, in which case, if only congested, bleeding will take place, or, again, by gently stroking it until its vessels are emptied; the vessels readily refill if the bowel is viable. (Power.)

These points cannot be too seriously considered, as at this stage of the disease we almost invariably find marked prostration, toxemia, etc., and only too frequently is the most insignificant surgical procedure too much for our little patients to withstand, therefore making it imperative that we should undertake the operation necessitating the least manipulation and the greatest chances for recovery.

Pringle, in 1899, suggested one of five methods of procedure in irreducible cases.

(1) Removal or excision of the whole invagination with end to end suture or other union.

(2) Removal or excision of the whole invagination with the establishment of an artificial anus.

(3) Leaving the invagination and establishing an artificial anus above it.

(4) To short circuit the bowel and let the invagination alone.

(5) Suturing the entering piece of intestine to the ensheathing tube at its neck by a continuous suture (other authors recommend an interrupted row of sutures), and then opening the ensheathing tube below the neck to extract the intussusception and to excise it within the sheath (or where possible from below), if accessible through the rectum.

(1) The first method is the ideal one and the end to end anastomosis, with simple suture, is in most cases the most satisfactory. The Murphy button shortens the time, but cannot be used in all cases. For instance, in the large intestine the appendices epiploicae make the two surfaces uneven and irregular. Power recommends packing with gauze about the bowel, with partial closure of the wound, where there is considerable shock and where there is a chance for the restoration of the circulation.

(2) The second method of artificial anus leaves the operation incomplete, and should only be resorted to in exceptional cases because of the high mortality.

(3) The third method almost invariably results in a permanent fecal fistula, while the gangrenous bowel, remaining in the abdomen, tends to promote further sepsis.

(4) The last objection also applies to this method.

(5) The fifth method proposed by Rydgier is apparently the most practical, but also has its drawbacks in such cases where there are strong adhesions between the invaginated bowel and the returning layer, also a danger of leakage along the thickening mesentery.

Dangers of Incomplete Operation. Before closure of the abdomen the operator should satisfy himself, with the least possible manipulation, that there are no remaining invaginations or other pathologic conditions which are resulting in obstruction of the bowel, or may cause a recurrence.

Summary of Treatment. (1) Intussusception demands an early diagnosis and immediate treatment.

(2) Abstinence from all food: far more important, purgation must absolutely be prohibited. The question of sedatives in the form of opium, etc., must rest with the physician.

(3) Irrigation may be tried once or twice under the proper conditions and in properly selected cases.

Conditions:

(*a*) Preparation for immediate laparotomy in case of failure.

(*b*) Complete anesthesia.

(*c*) Hot salt solution or plain water may be used under a pressure of not more than three feet, the fluid being allowed to remain in the bowel not less than ten minutes.

(4) Contraindications to irrigation.

(*a*) Recurrence after a previous complete or partial reduction.

(*b*) The very acute and severe types of this disease, which result

in early destruction of the bowel wall, but which cases are fortunately not the most frequent type.

(*c*) Where there are signs of beginning gangrene or ulceration, evidenced by subnormal temperature, profound toxemia, and other septic symptoms.

(*d*) Enteric intussusceptions.

(5) Laparotomy should follow failure of irrigation without delay.

(*a*) Attempted simple reduction from below upward.

(*b*) In irreducible cases. Resection within the bowel in selected cases, or, where this is not feasible, resection with end to end anastomosis should be attempted where the patient's condition makes it practicable, as an artificial anus or simple packing about the bowel requires a secondary, and only too frequently fatal, operation.

TAY-SACHS DISEASE IN TWO SINHALESE CHILDREN. C. C. de Silva and G. E. Tennekoon. (Brit. M. J., 2:768-770, Sept. 24, 1955).

Two cases of cerebromacular degeneration of Tay-Sachs type are described in Sinhalese boys, aged 2½ and 2 years when admitted to the Children's Hospital in Colombo, Ceylon. The children showed mental retardation, myoclonic fits, blindness with a dark purple patch at the fovea centralis surrounded by a white halo of avascular degenerated tissue, and primary optic atrophy. In one patient, the disease started at 2 months of age and the duration of the disease was two years and eight months; in the other patient, the disease started at the age of 2 years and its duration was only two months. Autopsy was performed in both cases and microscopic examination of the brain revealed swollen ganglion cells with a foamy cytoplasm and a small nucleus, which had been described previously by other workers. The only unusual feature in one case was the presence of large hyaline masses undergoing calcification in some parts of the brain substance and in the piarachnoid. The nature of these changes is unknown. The infantile type of Tay-Sachs disease has been described by other workers in Swiss and Norwegian children, and there is no longer any reason to state that Tay-Sachs disease arises almost exclusively in Jewish infants.—*J.A.M.A.*

DEPARTMENT OF ABSTRACTS

MOORE, H.: ADVANTAGES OF PYRIDOSTIGMINE BROMIDE (MESTINON) AND EDROPHONIUM CHLORIDE (TENSILON) IN THE TREATMENT OF TRANSITORY MYASTHENIA GRAVIS IN THE NEONATAL PERIOD. (New England Journal of Medicine, 253:1075, Dec. 15, 1955).

A case of transitory myasthenia gravis in a newborn infant, demonstrating the urgency of correct diagnosis and prompt treatment, is reported. The use of Mestinon proved to be of great help because it produced fewer of the undesirable muscarinic side-effects that were alarming while neostigmine was being given. Tensilon helped determine the proper dose of Mestinon. In the past, women with myasthenia gravis have been warned not to become pregnant. The use of these drugs so greatly facilitates the management of such an infant that women with myasthenia gravis can expect a greater chance of survival for their offspring.

AUTHOR'S SUMMARY.

SALK, J. E.: POLIOMYELITIS VACCINE IN THE FALL OF 1955. (American Journal Public Health, 46:1, Jan. 1956).

The discussion of vaccine safety contained in this report may be summarized as follows. The safety test has a twofold purpose: (1) as a test of a given lot of vaccine, and (2) as a test of the manufacturing process. Since the information on the lot in question gives a positive answer only with respect to a relatively small fraction of the entire lot, the answer to the question of the safety of the remainder of the lot from which the sample is drawn is had from the results of the test of the manufacturing process. While the safety of a batch of vaccine is determined by the presence or absence of infectious virus in a sample submitted to test, safety of a much higher order of magnitude is assured by a continuing examination of the manufacturing process as reflected by consistency of performance. The discussion of vaccine effectiveness reiterates the concept of relationship to antigenic mass and this as a function of potency and dose as well as spacing between inoculations. Data on the influence of age upon antigenic response are also presented. The question of future developments is discussed briefly and reasons given why these will be of technologic detail rather than of fundamentals.

AUTHOR'S SUMMARY.

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